MAD COW DISEASE
A GLOBALISED DISASTER

Britain's deception and delayed action over the BSE/CJD infection path have created animal and human health crises which are affecting much of Europe and are now spreading to the rest of the world.

by Lynette J. Dumble, PhD, MSc

International Co-ordinator
Global Sisterhood Network
E-mail: ljdumble@connexus.net.au
Website:
http://home.vicnet.net.au/~globalsn

BACKGROUND TO THE CRISIS

Since the 1996 admission[1] that British cattle suffering from bovine spongiform encephalopathy (BSE or "mad cow disease") had introduced the agent of an invariably fatal brain illness (a variant of Creutzfeldt–Jakob disease, referred to as vCJD) into the human food chain, BSE has subsequently spread to cattle in Austria, Belgium, Denmark, France, Germany, Italy, Ireland, Liechtenstein, Luxembourg, The Netherlands, Poland, Portugal, Spain, Switzerland and perhaps Sweden.

The report from the government-convened Lord Phillips Inquiry[2] into BSE in Britain claims that contaminated animal feed the size of a peppercorn can transmit BSE to cattle. To date, almost 200,000 cattle are known to have contracted BSE. Most cases have been in Britain, where the number exceeds 180,000. Another five million cattle, aged less than 30 months and without physical signs of BSE, have been slaughtered in preemptive measures to safeguard public health.

Since the initial 10 cases of vCJD were linked to BSE-infected beef in 1996, the disease claimed a further 90 human lives by February 2001, the vast majority of victims being permanent or temporary residents of Britain. Estimates of the anticipated number of human deaths as a result of BSE in cattle vary considerably, but, since the Lord Phillips Inquiry, the Blair Administration has warned that, over time, the figure could reach 250,000[3]—a big jump from the previous estimate of 136,000 vCJD deaths. The November 2000 announcement gave notice that UK authorities are working with the "worst case scenario" of one in every 250 people in Britain dying from the disease.

When questioned, microbiologist Dr Stephen Dealler explained that the revised figure was based on a "guesstimate" that the average Briton had probably eaten BSE-infected meat on 50 occasions, but he also admitted this: "At the moment, the number of cases of CJD we are seeing are doubling every year. If they double for a long time, then the numbers are in millions; if they double for just a few years, then the numbers are in thousands. At the moment, it is very difficult to know."

Unlike scrapie—the sheep equivalent of brain illnesses like BSE in cattle and CJD in humans which has been around for more than two centuries—BSE was unheard of until 1986, a decade after British cattle began to be fed the protein-rich remains of scrapie-infected sheep to accelerate their growth, and, coincidentally, four years after Britain commenced to expose cattle to organophosphate pesticides.

Human spongiform encephalopathy, or CJD, is also a disease of the 20th century, unknown until two German physicians, Creutzfeldt and Jakob, independently reported the first cases in the 1920s.

The agent of spongiform encephalopathies incubates in animals and humans for a prolonged period of time before outward signs of the infection become obvious: in cattle, after five years, on average; and in human cases of CJD resulting from human pituitary growth and infertility hormone injections or contaminated surgical materials, after as few as two to as many as 40 years.

By the time symptoms appear, the agent of BSE/CJD has already turned the brain into the sponge-like mass which led this group of diseases to be classified as spongiform slow virus disorders in the first instance. BSE-symptomatic cattle are left confused and trembling, deprived of their own feet to stand on, a furnace their tragic fate. CJD-symptomatic humans also suffer gait problems, and over varying periods of time—in some cases, weeks; in others, months and sometimes years—are progressively robbed of their every means communication, the ability to hear, see and speak. Gone, too, is their

understanding of written and spoken native language and, with this, every scrap of dignity.

In contrast to sporadic CJD, which has a natural incidence of less than one per million and generally claims victims who are in their sixties and seventies, BSE-related CJD was initially anticipated to be a disease which claimed the lives of much younger victims: teenagers and those in their 20s and 30s. Like the many other postulates responsible for clouding the BSE catastrophe with uncertainty, this theory fell apart in late 1999 with the vCJD death of a 74-year-old man, and led to questions of whether the CJD-like symptoms of Alzheimer's disease, which account for misdiagnosis in 10 per cent of sporadic CJD sufferers, might also lead, or have already lead, to vCJD escaping recognition in the elderly.

A BSE-infected food chain amounts to far more than the T-bone and rump steak, roast sirloin and hamburger which frequent the menus of today's predominantly meat-eating society. Taken together, these are the tip of an iceberg that has laid bare the vast array of bovine-based products which have come to be part and parcel of everyday lives. Coming quickly to mind are milk, cream, cheese, medical vaccines, health supplements and confectionery containing gelatin.

Equally, the public health implications of BSE extend much further than the consumption of infected bovine materials. Laboratory experiments since the 1970s have shown that, compared with the blood route, the oral pathway is a relatively inefficient means of transmitting these types of diseases from one animal to another. Extrapolating from that data, vCJD may ultimately claim fewer men than women, since tradition in every world region places more women than men in the kitchen to risk knife injuries with BSE-suspect meat.

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Additionally, it is not unreasonable to conclude that a BSE-infected beef consumer is henceforth a living incubator of vCJD. Like asymptomatic carriers of blood-borne infections such as hepatitis and syphilis, asymptomatic carriers of the vCJD agent have the potential to transmit vCJD to recipients of blood transfusions and organ transplants.

Overall, in the context of a BSE-infected food chain and the likelihood of BSE-contaminated blood and organ transplant supplies, it is fair to say that BSE now looms as a pandemic that may put HIV/AIDS in the shade.

ROOTS OF THE ORIGINAL BSE OUTBREAK

A number of theories have been put forward to explain the 1985 outbreak of BSE in British cattle. A small but respectable body of opinion argues that both cattle and humans have been poisoned by the widespread use of organophosphate pesticides; but an even larger body of opinion considers that Britain's BSE resulted from 1980s changes to the manufacture of animal protein–enriched cattle feed.

From 1985, when a mysterious disease now known as BSE appeared in Daisy, a dairy cow from Kent, the annual number of BSE-infected cattle rose to 731 within the space of three years. By 1989, 400 new cases appeared each week, and, by 1992, 100 new cases appeared each day. Also in the 1990s, animal species other than cattle, fed on cattle-containing rendered or raw meat and bones, began to die of BSE and CJD-like brain illnesses. Among them were African antelopes and domesticated and captive wild cats.

• Organophosphate Pesticides

But first, the "organophosphate theory" of how it might have all began. Three years before the first case of BSE was reported in Kent, British authorities ordered the compulsory spraying of cattle with the organophosphate pesticide phosmet, in order to combat a plague of warble fly. Manufactured by Zeneca, a subdivision of the British chemical giant ICI, and originally developed by the Nazis, phosmet is a neurological toxin. Related to the infamous birth-defect drug thalidomide, phosmet was used exclusively in Britain between 1982 and 1990.

In 1996, Mark Purdey, an organic farmer from Somerset in England, suggested that organophosphate pesticides such as phosmet might be behind Britain's BSE and vCJD crises.[4] His largely neglected, but nonetheless plausible, theory argues that the exposure of the bovine embryo to high doses of lipophilic formulations of organophosphates might have acted to trigger the deformation of cattle prion protein and hence the onset of the BSE. Certainly the timing, distribution and dynamics of phosmet usage in Britain tie in with the outbreak of BSE.

In Europe, the agrichemical division of Switzerland's Sandoz leaked tonnes of another organophosphate pesticide into the Rhine in 1986, killing all aquatic life in the river from Basel to the North Sea. It took seven years to revive the river, but it can also be argued that the organophosphate spill has played a role in the BSE which subsequently afflicted cattle in Switzerland, France and Germany in the late 1990s.

Purdey had investigated the clusters of BSE in cattle, clusters of traditional or sporadic human CJD in Britain, as well as clusters of a similar illness, chronic wasting disease, in deer and elk in the Unites States. He discovered that high levels of manganese—the metal sprayed in high doses on cattle via organophosphate pest deterrents—was a common factor. He suggested that, in addition to activating a prion mutation in cattle, excess manganese might also intensify traditional forms of CJD, which could thereby explain vCJD appearing in humans several decades younger than sporadic CJD victims. According to Purdey,[5] funding for BSE–organophosphate research may never eventuate. "No one's prepared to admit it, because it would involve massive compensation. By keeping the causal agent as something mystified, no one's to blame." He could be right.

The transnational companies behind the pesticide trade exert enormous political influence, with the giant Syngenta AG now heading the distribution of organophosphate pesticides. Syngenta's rise to become the world number one in agrichemicals (herbicides, fungicides and insecticides), number two in seed treatment and number three in seed supplies has been nothing short of astronomical, and can be traced to the 1996 merger between the agrichemical divisions of two Basel-based companies, Sandoz and Ciba.[6] April 1999 saw the agrichemical divisions of the Swedish Astra AB and the British Zeneca Group PLC merge to give birth to AstraZeneca, but by December 1999 both AstraZeneca and the Basel-based duo, by then known as Novartis, had also merged to form Syngenta. Boasting to be "the world's

first global, dedicated agribusiness company", the Novartis–AstraZeneca empire notched up combined sales in 1998 of close to US$8 billion, and with its new Syngenta identity has effectively removed the organophosphate faces of Zeneca and Sandoz from public view.

• Animal Protein–Enriched Feed

And now to the more popular "infected animal feed" theory, one which claims that BSE emerged from the post–World War II British strategy adopted to increase the milk yield of dairy herds. In brief, cows were fed on protein-enriched pellets made from the meat and bones extracted from the animal carcasses that littered abattoir and boning plants and from the animal leftovers discarded by butchers, restaurants and knackeries. The carcasses of scrapie-infected sheep, and, between the years 1985 and 1988, those of BSE-infected cattle, found their way into the protein-enriched animal feed which initially turned cattle into carnivores and ultimately into cannibals.

During the rendering process, the carcasses were milled and then decomposed in large vats by boiling at atmospheric pressure or higher pressure to produce a liquid protein layer under a layer of fat (tallow). Once the fat was separated, the protein solution was dried into a meat and bone meal product. Overall, Britain's rendering plants are part of a huge industry that supplied animal protein–enriched animal feed to livestock farmers, to pet owners, and to zoos for a number of animal species held in captivity.

The rendering process was deregulated in the late 1970s when, as a cost-saving measure, fat-removing solvents were dropped from the decomposition menu and the "cooking" temperature was lowered. During the same era, rendering procedures in other countries also underwent similar changes.

Numerous other countries exposed cattle to organophosphate pesticides, but only British cattle were afflicted with BSE by the mid-1980s—a phenomenon put down to the large proportion of scrapie-infected sheep within the mix of rendered animal carcasses in Britain. Nonetheless, it took until 20 March 1996—the day on which the British Prime Minister John Major publicly admitted that BSE apparently had jumped the species barrier from cows to humans—for Britain finally to ban the export of animal meat-and-bone-meal feed.

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FROM CATTLE TO HUMANS

As the BSE epidemic escalated from the initial case in Daisy the dairy cow, so too did concerns about human safety.[7] By and large, measures to eradicate BSE and prevent potentially infected tissues from reaching the human food chain were slow to commence. It was not until July 1988 that Britain banned the practice of feeding cattle with meal containing the ground-up remains of cows, but, for the best part of the next eight years, authorities persisted with assurances that it was absolutely safe to eat British beef. Few will forget the then Minister for Agriculture, John Gummer, stuffing a hamburger into his five-year-old daughter's mouth to demonstrate his confidence!

But, from the time BSE-like illnesses began to emerge in zoo ungulates and domestic and wild cats, it became impossible to ignore the possibility that BSE might also cross the species barrier to humans from the consumption of beef or dairy products or from the occupational contact of farmers and dairy and slaughterhouse workers with cattle.

Additionally, although BSE was presumed to have originated from scrapie, and scrapie was not a human pathogen, mouse-adapted strains of scrapie were known to adopt an altered host range after passage through hamsters, to become transmissible thereafter to rodents.[8] Similarly, human strains of kuru or CJD did not transmit to ferrets or goats until passaged through primates or cats,[9] as, too, a bovine strain of BSE was converted in the laboratory from nontransmissible to transmissible to hamsters by passage through mice.[10]

By May 1990, the CJD Surveillance Unit was established, a move extended three years later to Europe, to detect possible changes in British and European CJD epidemiology. In 1995, the unit in Edinburgh was notified of CJD in three victims, aged 16, 19 and 29 years. The British cases may not have been the first BSE-related deaths in humans; five years earlier, three cases of CJD in young patients had been reported in Poland.[11] The neuropathology of all three British cases revealed amyloid plaques, a feature which occurs in only five to 10 per cent of sporadic cases of CJD. By December 1995, the CJD Surveillance Unit had been informed of 10 suspected cases of CJD in persons under 50 years of age. Some turned out to have suffered sporadic or familial CJD, others a non-CJD illness, but one case was that of a 29-year-old and the other a 30-year-old. Neuropathology subsequently confirmed that both had suffered CJD and, like the three unusually youthful 1995 cases, had extensive deposition of amyloid plaques.

In the first months of 1996, two further cases of CJD emerged in youthful victims, and both with amyloid plaque neuropathology. By then, a distinctive clinical syndrome had begun to emerge to assist in diagnosing vCJD: young age at onset, early psychiatric symptoms, prominent ataxia, absence of periodic electroencephalographic activity, and a comparatively prolonged illness. Two additional vCJD cases were confirmed by the end of February 1996, and a report[12] on a total of 10 cases concluded that an unrecognised variant of CJD, unique at that time to residents of Britain aged less than 45 years, was probably due to exposure to BSE infection in cattle.

The link between vCJD and BSE was subsequently proved by laboratory studies[13] which demonstrated the identical characteristics of the pathological agents isolated from BSE-infected cattle and human cases of vCJD.

Laboratory evidence has also indicated that, rather than being present within beef muscle, the agent of BSE finds its way into the human food chain via beef products which are contaminated by nervous system tissue. This could have happened as a result of the cranium-stunning instruments which are used to immobilise cattle before they are killed by exsanguination; or from the inclusion of paraspinal ganglia in cuts of meat, e.g., in T-bone steak; or from the presence of residual spinal cord and paraspinal ganglia tissue in the paste of mechanically recovered meat which, in the pre–mad cow disease era, was added to cooked meat products like meat pies, beef sausages and a number of canned meat products.

Up until December 2000, all vCJD victims, with the exception of three cases in France, had lived in or visited the UK, indicating

that their infection was derived from British beef or beef products. British beef could well be the source of vCJD in the French cases, too, since approximately 10 per cent of France's beef for human consumption is imported from the UK. A smaller but significant proportion of British beef is consumed in most European countries. Given the vast number exposed to Britain's BSE-suspect meat and the distinct possibility that significant numbers are silently incubating the disease, the potential for human-to-human transmission looms large, notably with respect to blood and organ donation.

Two neuroscientists from Yale University in the United States, Laura Manuelides and the late Eli Manuelides, illustrated way back in 1975 that injections of human blood taken from CJD victims had the capacity to transmit CJD across the species barrier to laboratory animals. The implications of the Manuelides's experiment evaded authorities for the better part of the 1990s, though Canadian authorities spent C$15 million in 1995 to withdraw pooled plasma, already in the process of being transfused to thousands across the country, on the grounds that it contained a donation from a man who had subsequently died of CJD.[14] Similarly, in 1996, New Zealand authorities bit the bullet under weight of public pressure and quarantined blood products which had been contaminated by a donation from a CJD-infected donor.[15]

The years-long incubation time preceding CJD symptoms increases the difficulty to link a blood transfusion recipient's CJD with a donor source, and it falls within the realms of possibility that secondary CJD in a transfusion recipient may appear months or even years in advance of the primary CJD in a blood donor, as happened in the case of CJD in a liver transplant recipient which was eventually traced to a CJD-like illness in one of the blood donors.[16]

In the UK, whole blood or blood products from donors who have later died of vCJD has already been administered to a large but incalculable number of recipients. As a consequence, Britain now imports all plasma for transfusion purposes, and all blood from UK donors is filtered to remove the white blood cells which are the most likely carriers of vCJD infectivity. A number of other countries, including Australia, Canada, Germany, Japan, New Zealand, Switzerland and the United States now exclude blood donations from anyone who has resided in Britain for six months or more during 1980 to 1996. Similar policies are pending with respect to organ donation, and in Britain regarding the reuse of surgical instruments, especially those used in neurologic and ophthalmic procedures.

FROM BRITAIN TO EUROPE AND BEYOND

Britain's first move to halt the spread of BSE came in 1988 when a ban was placed on the 50-year-old practice of feeding protein-enriched cattle, sheep and other animal remains to cattle. A year later, UK authorities reassured both national and international audiences that mad cow disease was under control, and at the same time gathered scientists from the world's major laboratories engaged in human and animal spongiform disease research and a number of respected neurovirologists in order to seek advice.

Unwittingly, the solutions put forward by the experts shaped the events which have effectively spread mad cow disease across the globe. All were sworn to secrecy, notably regarding the export of cows and contaminated feed worldwide. One of them, Dr Laura Manuelides, physician and Professor of Neuroscience at Yale University, proposed that the epidemic could swiftly be brought to a close with the immediate culling of infected herds. Britain's attitude to the Manuelides solution was, in her words, "penny-wise and pound-foolish", and her idea was dismissed on the grounds that compensation for the owners of the herds was financially out of the question.

From then onwards, the global spread of mad cow disease went into full swing. Britons were placed at further risk of vCJD when an estimated 700,000 BSE-infected cattle entered their food chain, chiefly because the animals' slaughter age, usually three years, was below the age at which they would show signs of BSE infection.[17]

Next, the duplicity of the British Ministry of Agriculture, Fisheries and Food, known as MAFF, exposed mainland Europeans to an unknown quantity of BSE-contaminated veal among the two million calves transported to European saleyards between 1990 and 1995.[18] MAFF also sabotaged a 1990 Brussels ruling designed to prevent the spread of BSE outside Britain, when it issued civil servants with secret orders to skip the computer vetting of calves designed to exclude BSE-infected animals.

French official Gilbert Castille had suggested back in 1990 that Britain ought to be asked *not* to publish its research results, saying "it would be better to minimise BSE by practising disinformation".

Britain was not entirely alone in the processes which effectively globalised mad cow disease. In September 1996, the French newspaper *Libération* revealed that a memorandum from French official Gilbert Castille had suggested back in 1990 that Britain ought to be asked *not* to publish its research results, saying "it would be better to minimise BSE by practising disinformation". In fact, rather than ganging up on Britain, Brussels, via Guy Legras, head of the European Commission's agricultural directorate, warned of the financial repercussions from a beef panic and hushed news of the BSE situation.[19]

In a moral sense, the globalisation of mad cow disease then worsened. As Britain's market for the animal protein-enriched cattle feed dried up in Europe between 1988 and 1992, debt-burdened Third World countries became the replacement marketplace for attractively low-priced BSE-suspect meat and the same animal protein–enriched pellets believed responsible for Britain's BSE problems. Some European countries—Belgium, The Netherlands, France—which had imported the British meal, routinely shipped some of it to the Middle East and North Africa in concentrated form, where, before being fed to cows, the meat-and-bone meal is mixed with other locally produced animal feed. It wasn't until 1996 that Britain finally banned the export of meat-and-bone meal.

Britain's defence today is that her agricultural and public health authorities had debated the propriety of allowing the country's feed industry to continue exporting meat-and-bone meal after banning the practice at home. In the end, the decision was left to veterinary authorities in the importing countries, the argument being that those officials had been adequately informed of the risks!

The globalisation story did not stop with European and Third World countries. Between 1988 and 1996, Britain, in its thirst for greater and greater market profits through hybrid strains, also exported 3.2 million live cattle to 36 countries, representing every

world region. It was claimed that the cattle were BSE-free, but over the years mad cow disease in imported British cattle has been reported not only in Europe but also in Canada, Oman and the Falkland Islands. EU members on the Continent also have exported millions of live cattle all over the world. In December 2000, Kuwait reported a suspected BSE case in a three-year-old dairy cow imported from an undisclosed European country.

Ultimately, the dumping of BSE-implicated produce, considered unfit for sale in Britain, will be recorded as another shameful chapter of British imperialism.

The French Minister for Agriculture, Jean Glavany, sees it exactly in those terms, and recently commented that "...morally, they should be judged for that one day. They even allowed themselves the luxury of banning the use of such feed [in Britain], while allowing it to be exported."

Mainland Europe takes the high moral ground on the globalisation of BSE, yet, even as Britain ceased exporting animal protein–enriched cattle feed, other European countries were shipping tonnes of their own and did not implement total export bans until January 2000.[20]

Already there are reports of vCJD-like illnesses in India, Korea, Pakistan, South Africa and Thailand. One thing is certain, as the World Health Organization and Dr Manuelides have recently underlined: the social and environmental costs of a BSE-contaminated food chain in developing regions will far outweigh the multibillion-dollar estimates of Europe's present BSE-related crises.

Authorities in Australia, like those in the United States, claim to have imported little or no live cattle, beef products or livestock nutritional supplements from the UK, and boast about not being at risk of BSE. History may well tell otherwise.

As a wartime mentality of panic buying has taken over in the face of Britain's current meat shortage, there are lessons aplenty stemming from the man-made BSE pandemic, not the least of these being the price for placing profit ahead of public welfare and animal integrity.

By and large, it may prove somewhat immaterial whether organophosphate pesticides or scrapie/BSE–infected animal feed triggered the BSE pandemic in cattle, because both have been spread to the four corners of the globe with gay abandon. Should organophosphates be the culprit, it remains a matter of time before BSE outbreaks resembling those in Britain occur worldwide. Should the protein-enriched animal feed be the culprit, Britain's rendering economy has practically made certain that no region will escape. ∞

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About the Author:
Dr Lynette J. Dumble, PhD, MSc, is a medical and environmental scientist and the international co-ordinator of the Global Sisterhood Network. She is a former professor of surgery at the University of Texas in Houston, and senior research fellow in history and philosophy of science at the University of Melbourne. Her article, "From Mad Cows to Humans: The Next Global Plague" was published in NEXUS 5/01 (Dec 1997–Jan 1998). Dr Dumble can be contacted by e-mail at ljdumble@connexus.net.au.